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Gluten and dyspepsia

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Celiac disease (CD) affects approximately 1% of the population. It is a disease that causes flattening of the epithelial lining of the small bowel and is characterized by (sub) total villous atrophy. The histological alterations in CD are graded according to the modified Marsh Classification from Marsh 0 (normal mucosa) to Marsh IIIc (total villous atrophy). Common symptoms include, but are not restricted to, malabsorption, diarrhoea, malnutrition, failure to thrive in children and abdominal pain. Some patients with CD have no gastrointestinal symptoms or are asymptomatic (1). CD is caused by intolerance to gluten and a strict gluten free diet is the only available treatment. In the lamina propria of the small intestine of patients with CD there are T cells that respond to gluten fragments bound to the disease predisposing HLA-DQ2 and/or HLA-DQ8 molecules and secrete proinflammatory cytokines. The diagnosis of CD is based on the combination of HLA typing for DQ2 and DQ8, serum determination of specific CD antibodies (IgA anti-transglutaminase (tTG) and IgA antiendomysium (EMA) antibodies), on the results of small bowel biopsies and on the improvement of the symptoms after adherence to a gluten free diet (1-3).

In this number of GHFBB, Rostami Nejad and co-authors report on the prevalence of CD in

patients with dyspepsia and on the value of histology of the small bowel and of determination of specific CD antibodies in the diagnosis of CD in these patients (4). To study this, the authors performed small bowel biopsies and determined total IgA and IgA tTG (or IgG in case of IgA deficiency) in 407 randomly chosen adult patients who underwent diagnostic upper gastrointestinal endoscopy for dyspeptic symptoms. CD was identified by histological alterations characteristic of gluten sensitive enteropathy and by consistent CD serology. Biopsy results were classified as absence of CD (Marsh 0) or suggestive of CD (Marsh I to IIIc). There were 26 (6.4%) cases with enteropathy, among them 12 cases with Marsh I and 4 with Marsh II. Thirty three (8.1%) of the patients had tTGA level more than 15 u/ml, considered as tTGA positive, 10 of them (2.5%) had abnormal histology (Marsh I-IIIc), including 5 patients with Marsh IIIa-c (1.3%). This last figure is in line with frequencies of CD previously reported among patients with dyspepsia (5-7). The authors discuss if the real frequency of CD in dyspepsia should be considered as high as 8%, as assessed by determination of specific CD antibodies, since they have been shown to perform better than histology in the diagnosis of CD (8).

Some strong points of the study of Rostami Nejad and co-authors are, among others, its prospective design, the random inclusion of the 407 patients with dyspepsia who underwent upper gastrointestinal endoscopy during the study and

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the completeness of the total IgA and tTG results in their patients. However, the study has also shortcomings.

The authors considered levels higher than 15 U/ml as tTGA positivity. This level possibly represents the cut-off of normality of the tTGA determination in serum in their laboratory. However, it is known that lower levels of tTGA are less indicative for CD than higher levels, and that in CD there is a correlation between the TGA levels and the degree of small bowel damage. So, it should have been useful if the authors had presented the data on the correlation between the tTGA levels and the Marsch classifications of their patients. This is especially important in the cases with Marsch I alterations, since in absence of specific CD antibodies, these lesions are almost never indicative of CD (7). In addition, it is also known that “false positive” levels of tTGA are almost always present at low levels and in absence of EMA. So, the addition of EMA determinations, especially in these patients with elevated tTGA, should have added strength to the study.

The authors rightly state that there is no a single perfect test to diagnose CD in its own.

One additional, but important, part of the diagnosis is the improvement of the symptoms after the start of a gluten free diet. In this contest, information over the improvement of the symptoms of dyspepsia in the studied patients after adherence to the diet should be important, especially if, as the authors suggest, CD screening should be done in all patients with these symptoms.

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